AMENDMENTS TO THE SPECIFICATION

Please replace the paragraph beginning at page 21, line 26 with the following rewritten paragraph:

-- One gene, CYP7, encoding cholesterol 7a-hydroxylase (and previously assigned to 8q11-12 using both mouse-human somatic cell hybrids and FISH (Cohen et al., 1992)) was mapped in this contig using a primerset that we developed on publicly available sequence data (see Table 3). The YAC contig was constructed in parallel with the screening data of Whitehead Institute/MIT Center for Genome Research. --

Please replace the paragraph beginning at page 43, line 26 with the following rewritten paragraph:

-- To extend our observation to a larger number of tumors, we applied a reverse transcription-polymerase chain reaction (RT-PCR) approach. RT-PCR amplifications using primers specific for CTNNB1 and PLAG1 were carried out with RNA from tumors CG368, CG588, CG644, CG752, CG753 and T9587, which all carry the recurrent t(3;8), and from tumor CG682, which carries an ins(8;3) (q12;p21.3p14.1). RNA from tumor CG580, which carries a t(8;15), was included as a negative control. PCR experiments resulted in the generation of PCR products corresponding to hybrid transcripts consisting of PLAG1 and CTNNB1 sequences, in seven out of seven t(3;8) tumors analyzed (Fig. 6). In tumors CG368, CG588, CG682, CG752, and T9587, PCR products of 509 bp (Fig. 6A, PCR product A) and 614 bp (Fig. 6A, PCR product B) were generated, whereas in tumors CG644 and CG753 only the PCR product of 509 bp was found. The PCR product of 509 bp (from NECAT-UP up to MV6) ~~is~~ corresponds to a hybrid transcript containing exon 1 of CTNNB1 and exons 3 to 5 of PLAG1. The PCR product of ~~605~~614 bp contains an extra 105 bp, which corresponds to the alternatively spliced exon 2 of PLAG1. It points towards the presence of a related isoform consisting of exon 1 of CTNNB1 and exons 2 to 5 of PLAG1. This was also confirmed by nucleotide sequence analysis of the PCR products. We were also able to demonstrate that the corresponding reciprocal fusion transcripts are expressed. In all tumors except CG682, a PCR product of 130 bp was generated corresponding to a fusion transcript consisting of exon 1 of PLAG1 and exons 2 to 16 of CTNNB1. In Tumor CG753, an additional PCR product was detected, corresponding to a fusion transcript consisting of exons 1 to 2 of PLAG1 and exons 2 to 16 of CTNNB1. This additional band was also observed but

with weak intensity in tumor CG644. All these results indicate that in CG644 and CG753, the 8q12 translocation breakpoints are located in intron 2, whereas the breakpoints of tumors CG368, CG588, CG682, CG752, and T9587 are located in intron 1. Interestingly, using 5' RACE analysis with the tumor CG580, which carries a t(8;15) translocation, we found that the breakpoint occurs also in the same region leading to a fusion transcript with ectopic fused to exon 3 of PLAG1. --